

the left side as compared with the right is in harmony with this idea, the pressure of the right artery upon the vein being probably the cause by which an inhibition of the development, normally resulting in the complete disappearance of the loop, has been produced. The adhesions in the great majority of cases occur at the level at which the crossing of the vessel takes place. At the same time it must be recognized that the pressure of the artery is not in itself the cause of the adhesions; the occurrence of isolated cases in the right vein and of occasional cases in the left vein considerably below the level at which it is crossed by the artery being evidence of an ulterior cause, which I take to be the embryonic loop. It may be added that the study of sections taken through two suitably preserved veins in the region of the adhesion showed no evidence of inflammatory or other pathological processes.

The accompanying table shows the sex, age, cause of death, and type of adhesion in the majority of cases observed. In a few cases the necessary data were lacking, and in some instances the data in the third column are wanting in precision, but are given as furnished.

ACUTE GLANDULAR FEVER.

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THE lack of consideration of acute glandular fever as an independent disease, having a distinct and characteristic entity, or as probably being an aberrant form of influenza nostras, typhoid fever, or some other acute infectious disease, and the prevalence of many cases of fever of 101° to 104° F., with daily remissions of 1° to 2°, in which no positive diagnosis has been made, was the stimulus to investigate acute glandular fever as a common malady, and claim for it consideration and a conspicuous place among the acute infectious fevers.

DEFINITION. Acute glandular fever is an infectious disease developing, as a rule, without premonitory signs, and characterized by slight redness of the throat, high fever, and swelling and tenderness of the lymph glands of the neck, particularly those behind the sternomastoid muscles. The fever is of short duration, but the glandular hypertrophy persists for from ten days to four weeks.

HISTORICAL. The disease was first recognized by Pfeiffer, of Weisbaden, in 1889, who called especial attention to it under the name of "Drüsenfieber." Since Pfeiffer's description of the disease cases have been reported by numerous authors, mostly in Ger-

many and France. Heuber, Dawson Williams, Comby, Donkin, and J. P. West have contributed largely to the literature of glandular fever, but the disease continues to be considered uncommon and insignificant among infectious diseases.

ETIOLOGY. It occurs endemically and sporadically, especially among children. Exposure to cold and damp is supposed to have some relation to the disease. Eighty per cent. of all cases reported since its recognition have occurred in children. Byers reports 33 cases, in which the youngest was thirteen months and the oldest twenty-five years old. F. Marsh Dolloway reports 4 cases in adults twenty-four to forty-two years of age. Neuman claims it is due to a streptococcic infection, the organism entering by way of the tonsils without producing any local lesion, while H. Koplik suggests infection may be by way of the thoracic duct. Alerand, in France, has investigated thoroughly and written extensively on the aetiology of infection through the nose and throat, and the relation to tuberculosis. It has been suggested that the disease may be a peculiar form of influenza.

SYMPTOMATOLOGY. The period of incubation is from five to seven days. The onset is usually sudden, the patient complaining of headache, pain in the abdomen, vomiting, anorexia, and probably chills. The bowels are constipated, but the patient may rarely suffer from diarrhoea; the tongue is coated, and the face flushed; the evening temperature is 102° to 104° F., while the morning temperature is a degree or a degree and a half lower. The pulse rate is 90 to 130. On the second to the fifth day the enlarged glands appear, and during the course vary in size from a pea to a pigeon's egg, are quite tender, but there is rarely any redness or swelling of the skin. The swelling of the glands persists for two or three weeks. In the severe cases headache is always complained of as an initial symptom, together with abdominal pain and vomiting. Byers, in his series of cases, found the throat unaffected beyond a slight congestion. Many of the patients complained little or not at all until the glands of the neck seemed suddenly to enlarge. The glands affected were the deep ones under and in front of the sternomastoid muscle. No suppuration occurred in any of the cases and the enlargement of the glands subsided in a week or two. The lymph nodes are hard, tender, freely movable, and form a regular chain around the neck. The axillary and inguinal glands are most always involved, the spleen is enlarged in 80 per cent. of the cases, and the liver in 90 per cent. Mesenteric glandular enlargement was found in 40 per cent. Convalescence is, as a rule, very rapid, and uninterrupted by complications.

DIFFERENTIAL DIAGNOSIS. *Acute leukemia* in the early stages closely resembles glandular fever. Differentiation is made by blood examination, the leukocytosis in leukemia being marked and persistent, 1 to 30 to 50 being the relation of white to red cells.

Anemia is pronounced and progressive, and the temperature of longer duration than in glandular fever. A differential leukocyte count shows the predominance of lymphocytes or myelocytes.

Syphilis is excluded by a history of initial lesion or lues in the parents, by marked anginose symptoms, and cutaneous eruptions, together with response to mercury.

Pseudoleukemia. In Hodgkin's disease the glandular enlargement is at first unilateral, and is accompanied by progressive emaciation with marked anemia, oedema, and late pyrexia, the fever usually being intermittent.

Tubercular adenitis is excluded by the presence of a tubercular history. The glandular involvement is usually localized and accompanied by redness and tumefaction of the skin, with suppuration. The fever of tubercular adenitis is of longer duration than that of glandular fever. By reaction to tuberculin or demonstration of tubercle bacilli in the sputum, the diagnosis is corroborated.

CASE I.—W. W., male, white, aged sixteen years; born in West Virginia; occupation, farm laborer; well nourished and developed. Brought to hospital on morning of August 31, after having been confined to house two days. Complained of headache, pain, and stiffness in neck, pain in abdomen, cough, and weakness. The temperature on admission was 104° F.; pulse, 96; respirations, 28. The patient gives no history of disease predisposed to by heredity. No history of prodromes preceding confinement to house, and was perfectly well two days previous to admission to hospital.

The face was flushed, expression calm, eyes bright, decubitus dorsal recumbent. The skin was dry and fine. The glands of the cervical region very slightly enlarged, the neck somewhat stiff and tender. No redness of skin above the glands. Tonsils, nasal and oral pharynx, slightly hyperemic. Examination of chest gives negative physical signs. The heart was normal, the pulse-rate 96, the pulse full. Blood pressure, 115 mm. The pulse declined gradually with the fever and remained of good quality and volume. Blood pressure, 130 mm. on fifth day.

Examination of Blood. Malarial parasite negative on the second and sixth days. Widal reaction negative on the second, sixth, fourteenth, and twenty-first days. Red blood count, 4,000,000; hemoglobin, 90 per cent.; leukocytosis, 14,000 on the sixth day, 8900 on the twenty-first day.

The temperature reached fastigium (104.6° F.) on the fourth day and persisted, with slight daily remissions of 1° to 2°, until the seventh day, when the temperature, declining by lysis, became normal and so remained.

Gastro-intestinal symptoms present were anorexia, fetid breath, tongue moist, swollen, and heavily coated, with reddened tip and edges. Spleen markedly enlarged. Liver slightly enlarged. Abdomen tender on pressure. Stools well digested. No tympanites,

distention, or diarrhoea present preceding, during, or following the disease. Slight dysphagia. Rose spots negative. The glandular hypertrophy, pain, and tenderness first noticed in the postcervical region later became evident to a less prominent degree in the axillary and inguinal regions. The mesenteric glands were never palpable. The pain and tenderness over the glands ceased with the decline of fever, but the glands remained discretely enlarged, though diminishing, at the time patient was discharged. Examinations of sputum for *Bacillus tuberculosis* proved negative on two occasions during the course of the disease.

CASE II.—S. F., female, white, aged twenty-seven years. Patient was taken ill with headache, anorexia, abdominal pain, and slight dysphagia. Temperature, 103° F.; pulse, 96; respirations, 26. Glandular enlargement became evident on the fourth day of disease. The glands were not painful, but slightly tender on pressure and unaccompanied by redness of skin or tendency to suppuration. The temperature became normal on the eighth day, and so remained; the glandular enlargement persisting for over three weeks. Convalescence was uninterrupted and not followed by prostration or anemia. Patient was kept confined to bed for five weeks after cessation of the fever because of glandular condition, although the general condition was good after the return of temperature to normal. Widal reaction negative on the eighth day; leukocyte count, 7000 on the eighth day. A tentative diagnosis of enteric fever was made in both instances, but was later abandoned in the absence of frank symptoms.

Glandular fever presents many of the characteristics of influenza, and may probably be associated with influenza as an aberrant form. If *Bacillus pfeiffer* should be found in the secretions, glandular fever should be considered associated with endemic influenza-nostros or la grippe, occurring sporadically as a protean type; but repeated efforts have not demonstrated this association. The highly contagious nature of influenza and pandemic invasions are absent in glandular fever. Splenic and hepatic hypertrophy are rarely present in epidemics of influenza occurring in the United States.

The absence of characteristic features of typhoid fever in these cases, as in a score or more which had come under my observation in the George Washington and Emergency Hospitals, led me to the conclusion that many of these cases are acute infectious diseases other than typhoid. The absence of prodromal symptoms, rose spots, typical pulse, Widal reaction repeatedly tested for, and the presence of glandular hypertrophy with pain and tenderness, the glands remaining enlarged after the cessation of the disease without suppuration, the short duration of the fever, with rapid convalescence, and the presence of leukocytosis during the fever tend to a diagnosis of some disease other than enteric fever. I believe acute glandular

	INFLUENZA.	ACUTE GLANDULAR FEVER.	LEUKEMIA.	TUBERCULAR LYMPHADENITIS.	TYPHOID FEVER.
Onset	Abrupt.	Abrupt.	Gradual.	Gradual.	Gradual.
Causes	B. Pfeiffer.	Unknown.	Unknown.	B. tuberculosis.	B. typhosus.
Fever	Intermittent or continued.	Continued.	Variable.	Intermittent and remittent.	Continued.
Nutrition	Temporarily impaired.	Temporarily impaired.	Progressive emaciation, weakness.	Emaciation, slight or extreme.	Temporarily impaired.
Weight	Loss followed by recovery.	Loss followed by recovery.	Progressive loss.	Progressive loss.	Loss followed by recovery.
Anginose symptoms	Usually marked.	Absent or very slight.	Absent.	Always present.	Variable.
Spleen	Normal.	Hypertrophied.	Hypertrophied.	Normal.	Hypertrophied.
Blood changes	Slight secondary anemia; slight leukocytosis.	Slight secondary anemia; slight leukocytosis.	Extreme secondary anemia; leukocytosis marked and persistent; marked increase of myelocytes or lymphocytes.	Secondary anemia.	Slight secondary anemia. Leukopenia.
Glands	Rarely involved; always confined to cervical region; never suppurate.	Discretely enlarged, painful, and tender; never suppurate; cervical, axillary, and inguinal glands involved; hypertrophy subaltes gradually; pain and tenderness cease with fever.	Markedly enlarged; no pain or tenderness other than produced by pressure; never suppurate; cervical, axillary, and inguinal glands involved; hypertrophy progressively increases, rarely subsides.	Cervical most commonly involved, usually localized; tend to suppurate and condesc.	Glandular involvement very rare.
Sputum and nasal secretions	B. Pfeiffer, probably.	Negative.	Negative.	B. tuberculosis, probably.	Negative.
Duration	Weeks.	Weeks.	Prolonged, months or years.	Months.	Weeks or months.
Widal reaction	Negative.	Negative.	Negative.	Negative.	Positive.
Gastro-intestinal disturbances	Vary; may be slight or severe.	Onset with vomiting; during course resembles typhoid.	Slight at first; more pronounced as disease progresses.	Negative.	Marked.
Glandular enlargement	Follows fever.	On the fourth or fifth day after onset of fever.	First evidence of disease.	Followed by fever.	Follows fever.
Convalescence	Gradual.	Rapid.	Rare or retarded.	Tardy.	Gradual.
Complications	Occasional.	Rare.	Rare.	Common.	Frequent.
Mesenteric enlargement	Never.	40 per cent. of cases.	Late.	Probable.	Rare.

fever to be a very common disease, unrecognized, and that it frequently is mistaken for aberrant forms of typhoid or influenza. From my observation and study of cases, I have become firmly convinced that some cases of enteric fever of short duration have been nothing other than acute glandular fever, and that this disease should be given a more prominent place than it has among acute infectious diseases, not only as occurring among children, but among adults as well. The annexed table may prove serviceable in the differential diagnosis.

EPIDEMIC CEREBROSPINAL MENINGITIS IN HARTFORD, CONNECTICUT, DURING 1904-1905.

WITH SPECIAL REFERENCES TO THE CASES TREATED AT THE
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CONNECTICUT has been visited by three previous extensive epidemics of cerebrospinal meningitis, which have been recorded with painstaking accuracy by the physicians residing in the towns where it was especially prevalent. The first epidemic, continuing from 1806 to 1816, is well known to the student of medical history in this country by the excellent little monograph which Elisha North,¹ then of Goshen, wrote upon it. Dr. Lemuel Bacon, in a manuscript which is still extant, has also discussed this epidemic as it appeared in Hartford. The second epidemic was seen in Middletown in 1823, and formed the subject of a forgotten admirable pamphlet by Dr. Thomas Miner, who called the disease typhus syncopalis.² He had previously, also, referred to it in a book, which he and Dr. William Tully wrote, entitled *Essays upon Fevers and Other Medical Subjects*.³ The third epidemic occurred in 1873, and formed the basis of a paper by Dr. Horace S. Fuller, of Hartford, which, unfortunately, has never been published. The epidemic we shall consider began in March, 1904, and continued, though in a much abated form, until December, 1905.

¹ A Treatise on a Malignant Epidemic Commonly Called Spotted Fever, New York, 1811, pp. 250.

² Typhus Syncopalis, Sinking Fever, or the Spotted Fever of New England, as it Appeared in the Epidemic of 1823, in Middletown, Conn., Middletown, 1825, pp. 48.

³ Essays on Fevers and Other Medical Subjects, Middletown, 1823, p. 484.